

Long-term use of potassium perchlorate

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Summary

A case of Graves' disease with potassium perchlorate for 22 years without ill effect is described. Thyrotoxicosis recurred 4 weeks after the medication was withdrawn, suggesting that euthyroidism had been maintained by chronic use of the drug. As toxicity of perchlorate is probably dose related, it is suggested that long-term use of low dose perchlorate may be no more hazardous than alternative antithyroid therapy.

Introduction

Potassium perchlorate was extensively used as an antithyroid agent in the late 1950s and early 1960s (Crooks and Wayne, 1960): by competitive inhibition of the trapping of iodide by the thyroid it was effective in reducing thyroid hormone production by the gland, and consequently in relieving symptoms of thyrotoxicosis (Godley and Stanbury, 1954). No evidence has been produced to suggest that it might influence the natural course of thyrotoxicosis. Following reports of toxicity, in particular of bone marrow hypoplasia (Barzilai and Sheinfeld, 1966) it fell into disfavour, and is now used mainly for investigative purposes. The author now reports a case of long-term use of potassium perchlorate.

Case report

A 72-year-old female was referred to the Thyroid Clinic in August 1980 with symptoms of thyrotoxicosis. She had undergone a partial thyroidectomy in another hospital in 1945 for thyrotoxicosis. In 1956, she was diagnosed as suffering from pernicious anaemia, and started on regular vitamin B₁₂ therapy. In 1958, her thyrotoxicosis recurred both clinically and biochemically. She was rendered euthyroid with potassium perchlorate, one g/day by mouth for one month, and maintained thereafter on 200 mg/day, with good control of symptoms. She remained clinically euthyroid on this therapy without ill effect until May 1980, when her GP stopped the potassium perchlorate. Four weeks later she developed symptoms of thyrotoxicosis, including weight loss, heat

intolerance and excessive sweating, and was referred to the Thyroid Clinic. Apart from pernicious anaemia affecting a maternal aunt, she gave no other history of note.

On examination she was clinically thyrotoxic, with warm moist palms and hyperkinetic movements. She had a tachycardia of 120 beats/min. A small diffuse goitre was palpable, with the left lobe being larger than the right; no bruit was audible. There was no ophthalmopathy. Other examination was unremarkable.

Initial thyroid function tests confirmed the clinical impression with a T₄ of 245 nmol/l (normal range 59-174), T₃ of 4.2 nmol/l (normal range 1.29-3.3) and a free thyroxine index of 77.4 (normal range 17.8-46.1) (Amersham radioimmunoassay kit). Thyroidal uptake of ¹²³I at 20 min after i.v. administration of the tracer was elevated at 9.7% of dose (normal range 2-8%); the precipitin test for thyroglobulin antibody was negative. A technetium scan of the thyroid showed a diffuse uptake of isotope, with the left lobe more active than the right. Haemoglobin was 11.3 g/dl with an MCV of 88 fl; WBC was $5.3 \times 10^9/l$, with normal film appearances; platelet count was normal at $194 \times 10^9/l$.

A diagnosis of Graves' disease seemed reasonable in view of her history of pernicious anaemia, and the diffuse thyroid scan appearance. In view of her age, and recurrent nature of her illness, she was treated with radioactive iodine (¹³¹I) by mouth, and is now clinically euthyroid.

Comment

This appears to be a unique case with maintenance of euthyroidism by the use of potassium perchlorate over a period of 22 years. The temporal relationship between withdrawal of perchlorate and recurrence of thyrotoxicosis suggests that perchlorate was responsible for the maintenance of euthyroidism, by continued chronic intrathyroidal iodine depletion. With the withdrawal of perchlorate, unblocked excessive iodide trapping was able to occur, leading to excessive thyroid hormone production. This

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situation is analogous to the cases of thyrotoxicosis 'unmasked' in populations by the introduction of dietary iodine supplementation (Connolly, Vidor and Stewart, 1970).

This patient received potassium perchlorate for 22 years without any untoward effects. The reports of adverse reactions to potassium perchlorate suggested that these effects of the drug were dose related (Morgans and Trotter, 1960), and it may be that low dose perchlorate (200 mg) is no more toxic than the current generation of thiourylene antithyroid drugs (Barzilai and Sheinfeld, 1966).

References

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